STUDIES ON DYSENTERY

A PRACTICAL SURVEY OF ONE THOUSAND CASES IN A GENERAL HOSPITAL IN EGYPT, 1918 - 1919.

A THESIS

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STUDIES ON DYSENTERY.

INTRODUCTION.

During the latter part of the Great War it was my privilege to serve in the Royal Army Medical Corps, with the 54th Division at Tel-el-Kebir, Egypt. Attached to No.7 General Hospital I had sole charge of five hundred beds which had been specially allocated for cases of dysentery. The patients under my care comprised British troops from Mesopotamia and the Palestine front, and a large number of prisoners of war, Germans, Austrians and Turks.

I was stationed at this Hospital from June 1918 to August 1919, dealing with cases of dysentery all the time. I propose therefore that/

that the main part of this Thesis shall be devoted to an account of the observations which I was then enabled to carry out.

The investigation consisted of a study of a thousand cases of dysentery, admitted to my wards during 1918 and 1919. Owing to the urgency of the medical situation in Egypt at that time, my routine duties naturally did not allow of a concen-:trated research into the subject of dysentery, but observations based on this series of cases were made on the pathology, diagnosis and treatment of the disease.

The pathological studies which formed part of my daily work were taken up mainly with examination of the stools and post-mortem examinations. This part of my Thesis therefore, although couched in somewhat general terms, is based on a careful study of several hundreds of stools, and on over twenty autopsies.

Opportunity/

Opportunity was given me also to study the effect of emetine bismuth iodide on carriers of amoebic dysentery, and of anti-dysenteric serum in acute bacillary dysentery. Both these methods of treatment at that time were more or less on trial, and my results may therefore claim to have a certain clinical significance.

I was the first to introduce chlorine water lavage, as an adjunct in the treatment of dysentery, into the routine hospital practice of the Army Medical Corps in Egypt. My results were certainly encouraging, and I had the honour to be mentioned in a special report submitted by the Senior Medical Officer to the Director of Medical Services. This form of therapy was adopted by a large number of hospitals in the Eastern War Zone and was in part incorporated in the Training Manual of the Royal Army Medical Corps.

This/

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I. HISTORICAL SURVEY.

HISTORICAL SURVEY.

Our real knowledge of dysentery is of comparatively recent date. Although Hippocrates was the first accurately to describe the disease which we now know as dysentery, yet there is good ground for believing that the disease existed in Egypt and India for centuries before Christ. Many of the older writers failed to differentiate conditions which showed blood and mucus in the stools from those with blood alone. And it was only during the last century that authorities have considered the associ-:ation of mucus with the blood as an essential in clinical diagnosis.

The etiology and epidemiology of bacillary dysentery have been fairly definite since about 1900; amoebic dysentery, on the other hand, was not in such a secure position till about 1913.

A/

A brief historical note, therefore, may be of in-:terest.

In 1752 Sir John Pringle published one of the earliest modern descriptions of dysentery, in his work, "Observations on the Diseases of the Army", based on his experiences in Flanders. "The disease was worse in fixed camps at the end of the close, hot summer and in the autumn, when it was epidemic and contagious". He looked upon "heat and moisture of the air" as the remote causes, and a "putrefying state of the blood and scurvy" as predisposing ones. He adds the following inter-:esting remark; "but having since perused the curious dissertation by Linnaeus, in favour of Kercher's suggestion of contagion by animalculae, it seems reasonable to suspend all hypothesis till that matter is further inquired into". Over a century elapsed before this prophetic writing was fulfilled.

Annesley (1828) published his, "Researches on the Diseases of India", containing a full/

full description of dysentery and abscess of the liver as seen in Madras. He found liver abscess post-mortem in twenty-six out of fifty-one cases, and it is therefore probable that he was dealing chiefly with the amoebic variety of the disease. Sir Leonard Rogers says, "Annesley's classical description is by far the best account of amoebic dysentery to be found in the older writings, if indeed it has ever been surpassed".

Twining (1835), Parkes (1846), and Mackinnon (1848) all described fully the disease in various parts of India, and Baly in his Goulstonian lectures of 1847 gave a good description of dysentery in the Millbank Prison in London. The type he met with was clearly the bacillary form, and he con-:sidered that the outbreaks were "due to a local influence, malarial in nature, arising from the soil, and that it was not due to bad water, inadequate ventilation, or sewage defects".

Woodward/

Woodward (1880), in his, "Medical and Surgical History of the War of the Rebellion", gives a very exhaustive account of diarrhoea and dysentery as it occurred in the United States of America.

Fayrer (1881), Maclean (1886), Chevers (1886), Kelsch and Kiener (1889), and numerous other writers have also given us excellent descriptions of the disease in various parts of the world. All these observers have shown that bacillary dysentery has always been the prevalent type of the disease in temperate climates, occurring especially in institutions and in military camps during times of war. In warm climates the pre-:valent type would appear to have been the amoebic variety, although bacillary cases were also met with.

It is hardly surprising that amoebic and bacillary dysentery were not differentiated from each other by these early workers, as the clinical symptoms/

symptoms of the two forms are so often indistinguish-:able. In 1859 Lambl first discovered "amoebic" forms of the organism in the human intestine, but controversy continued as to whether the organisms were the active causative agent in the production of the disease, or merely accidental or secondary concomitants.

In 1898 Shiga discovered the Bacillus dysenteriae, and this, with other closely allied strains of the coli group, was proved to be the cause of the forms of dysentery which prevail in temperate climates as well as of a proportion of tropical cases. And not till about this time did the Amoeba Dysenteriæ come to be recognised generally as the agent in pro-:ducing a large proportion of cases of dysentery in the tropics as well as in sub-tropical climates, and the discordant facts recorded by research workers in all parts of the world became reconcilable.

It may be of interest to recapitulate very/

very briefly an outline of previous work on the treatment of dysentery. Sir John Pringle in 1774, when confronted with epidemic bacillary dysentery during the Walcheren Expedition, recommend-:ed moderate bleeding, careful purging, opium,and ipecacuanha in hourly doses of five grains.

Annesley (1828) also advocated bleed-:ing, warm baths, calomel and opium. He appears only to have given ipecacuanha in one to three grain doses once or twice a day.

Other early workers, Twining (1835), Parkes (1848), Baly (1847), Maclean (1886), all gave full attention to the treatment of symptoms and had considerable success in the administration of ipecacuanha by the mouth.

Ipecacuanha, or Brazilian root, was first brought to Europe in 1658 by Piso and given in the form of an infusion or decoction. It appears to have been used in India as early as 1660, but later/

later to have fallen out of use, both there and in Europe. Annesley and Twining, in the early part of the nineteenth century employed it as we have seen, and with significant success. But Docker is generally credited with having successfully re-introduced the drug in 1858, amongst British troops in Mauritius.

It is interesting to note that while ipecacuanha was so signally successful in certain cases these observers noticed that many cases showed no improvement whatsoever. Now of course, we know the cases to have been bacillary in origin . And in such cases calomel and opium were the drugs most generally used. Maclean (1886) writes: "How ipecacuanha, almost deserving the name of a specific, came to be superseded by calomel and opium in the treatment of dysentery in the East is one of the most curious questions in the history of tropical medicine". The differentiation of the amoebic from/

from the bacillary type of dysentery has furnished the solution to Maclean's problem.

Emetine, isolated by Pelletier in 1817, was used for the first time in the treatment of this disease by Bardsley (1829) and Tull-Welsh (1891), but its importance as a specific therapeutic agent in the treatment of amoebic dysentery was not fully recognised until Leonard Rogers reported its success in 1912.

At the time when the main part of this work was carried out, during the last two years of the Great War, ipecacuanha and emetine, administer-:ed orally and by injection, were both recognised as of undoubted value in amoebic dysentery. And the bacillary forms were found to respond fairly well to specific therapy. During the past twelve years immense steps have been taken in the treatment of the dysenteries, but it yet remains to be seen whether in times of war our modern chemotherapy and bacteriophage/

bacteriophage treatment will enable us to combat even more successfully the "dreaded scourge of the camp".

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II. A PATHOLOGICAL STUDY OF DYSENTERY, WITH SPECIAL REFERENCE TO CYTO-DIAGNOSIS.

A PATHOLOGICAL STUDY OF DYSENTERY WITH SPECIAL REFERENCE TO CYTO-DIAGNOSIS.

PATHOLOGY OF AMOEBIC DYSENTERY.

Amoebic dysentery is due to infection by the Entamoeba histolytica, and by it alone. No other amoeba is capable of causing this disease, and it requires no help from secondarily infecting organisms of the bowel in the production of its lesions.

The/

The life-history of this parasite is briefly as follows. The encysted form is swallowed with the food. Arrived in the small intestine, the digested juices there dissolve the capsule and set free the small daughter amoebae, which make their way to the large intestine and thence down the lumina of the mucosal glands into the submucosa. There they grow into the adult or vegetative form, multiply by binary fission, and give rise to a direct colliquative necrosis of the submucosa and muscularis mucosae by means of a proteolytic ferment which they are able to secrete.

Direct communication with the lumen of the gut - i.e., ulceration - is only attain-:ed when either the solvent attacks the intercellular cement of the mucosa or the necrosis extends far enough to interfere with its blood-supply. According to which occurs first the mucosal cells are shed singly or <u>en bloc</u>, and an irregular ulcer with ragged,/

ragged, undermined edges is formed. The loss of substance is far greater in the submucosa than in the mucosa, as large vegetative amoebae are found in the greatest numbers there; often sinues lead from one ulcer to another under bridges of perfectly healthy mucosa.

As the amoebae approach the surface of the mucous membrane again they divide more and more rapidly, and consequently become smaller and smaller until on the surface of the gut they assume the "minuta" form. Many of them now encyst, and they are passed out of the body in the stools as cysts or E. minutae. It is worthy of note that the E. minutae produce no destruction of the tissues, and consequently no symptoms of dysentery; a fact which is distinctly in favour of the larger, or older, amoebae producing a proteolytic ferment.

Adami/

Adami was the first to point out that the reproductive and the higher functional activities of cells are mutually antagonistic; the more em-:bryonic the cell the more readily does it multiply, and thus the more rapid is the growth of a neoplasm composed in greater part of cells of embryonic type. The smaller size of these amoebae is due to their more rapid division, which has used up all their re-:serve of energy; consequently none is left for the development of that particular function which differ-:entiates E. histolytica from all other amoebae.

It has never been shown that the entamoebae produce a diffusible toxin, or that they are capable of exerting any poisonous effect other than the proteolysis of cells in their immediate neighbourhood.

In consequence, the onset of the disease as a rule is insidious - it may be even unnoticed/

unnoticed - and it is not until a comparatively large area of the gut is involved that the patient becomes at all acutely ill. It will thus be noted that the mucosal cells are in general healthy, apart from the areas of actual infection, while at autopsy the typical picture seen is one of areas of circumscribed necrosis, dotted like islands in the midst of healthy pink mucosa.

Since emetine treatment was universally adopted, cases where the bowel has undergone "gangrene en masse" are, fortunately so rare as to warrant only passing notice. This condition is brought about by an extensive amoebic invasion of the submucous and muscular coats of the intestine, with consequent thrombosis of blood-vessels, and the blood supply of the bowel is cut off as effectually as by multiple thrombosis arising from any other cause.

The result is that the entire thickness of the bowel over a greater or lesser extent becomes gangrenous,/

gangrenous, and is passed as stringy black sloughs having a horribly foetid odour. On very rare occasions it has even been impossible to remove the colon at autopsy, as all that remained to represent it was a slimy, stringy, black mass lying in a bath of corruption. Unless one had actually seen such a case it would be inconceivable that a man could survive long enough for his intestines and his peritoneum to attain such a state.

It has frequently been shown that fatal cases of amoebic dysentery often terminate with a blood infection by members of the Streptococcus faecalis group. There is nothing astonishing in this if one considers how often the intestinal bacteria invade the body before death in different chronic and subacute diseases. The astonishing part is that it does not occur more frequently in amoebic dysentery - a disease in which there is such an extensive solution of continuity of the mucosal barrier. The point which the writer would/

would like to emphasise is that in the vast majority of cases of non-fatal amoebic dysentery no such in-:vasion of the blood stream can be demonstrated by blood culture.

This may be due to one or two causes acting together or separately. In the first place, the liver may act as an efficient bacterial filter; in the second, the bowel itself is probably endowed, even when diseased, with a high immunity against just such organisms. Wright has shown that different parts of the body possess different degrees of resisting-power against pyogenic organisms. It seems reasonable to suppose that this local im-:munity acquired by countless minute inoculations with the bacteria-containing fluid in which the mucosa has been bathed since birth is sufficient to protect not only the body against invasion by these bacteria, but even the mucosa itself when the latter is/

is partially destroyed by amoebae.

In amoebic dysentery only when extensive areas are killed by interference with their blood supply does any secondary infection of the mucosa take place, just as a limb may become affected by wet gangrene after the blocking of its blood supply. When, however, a pathogenic organism, such as a dysentery bacillus, which is a stranger to the mucosal barrier, and against which the mucosa is in consequence not immunised, arrives on the scene in sufficient numbers and virulence, immediately its destructive properties come into play.

When sections are made through the edges of early amoebic ulcers, and more particularly when the unbroken amoebic nests in submucosa or liver are examined histologically, the observer is struck by the absence of the usual cellular infiltration characteristic/

characteristic of inflammatory reaction.

This is all the more noticable when a comparison is made with the corresponding sections of early bacillary dysentery which is an inflammatory disease.

Again, the so-called amoebic liver "abscess", when sterile, is not an abscess at all. It is a primary colliquative necrosis of hepatic tissue, which is broken down and changed by a pro-:teolytic enzyme from protein to albumoses and pep-:tones; the patient's symptoms are due to the absorption of these poisons directly into the blood stream and can be reproduced experimentally by the intravenous injection of peptone.

To sum up: the pathology of histo-:lytica amoebiasis is a primary degeneration due to the/

the chemical digestion of the cells in the immediate neighbourhood only of the amoebic enzyme. It is not a primary inflammation with consequent degenera-:tion; any inflammation which occurs is due to secondary microbic infection, is generally not severe, and takes place late in the disease.

The nature of the cellular exudate in the stools and in the uncontaminated liver "pus" is in entire accord with this basal fact of its path-:ology.

PATHOLOGY OF BACILLARY DYSENTERY.

Very different from the amoebic lesions are the changes which occur in bacillary dysentery. The/

the infecting agents belong to a different kingdom.

The two diseases are so totally dis-:tinct that the only excuse for grouping them both under the heading of "Dysentery" is that of historical usage. In logic it were as reasonable to group cancer and tuberculosis of the intestine under the same heading because they may both give rise to what used to be called the "bloody flux".

Whatever may be the route of infection the essence of the pathology of bacillary dysentery is the intense diffuse catarrhal inflammation of the mucosa, which according to the severity of the attack may resolve or may go on to degenerative changes, such as coagulation necrosis and sloughing. It is a primary inflammation of bacterial toxic origin, with all the cardinal signs of this condition - viz., "rubor, tumor, calor, dolor et functio laesa".

The process is essentially one of diffuse destruction of the superficial layers of the mucosa, accompanied by oedema, large immigration of leucocytes, diapedesis of blood cells, and haemorrhag-:ic extravasation - such as is seen in all cases of acute bacterial inflammation of tissue. The immigration of polymorphs is an especially note-:worthy feature. At autopsy the appearance of the intestine in any case of bacillary dysentery will vary with the intensity of the infection and the length of time the patient has survived it.

In the so-called fulminating ileo-colic dysentery, the entire surface of the lower part of the ileum and large intestine, excepting Peyer's patches and the appendix, is seen to be the site of intense inflammatory change. No part escapes, as it does in amoebic dysentery; the bowel wall is oedematous, enormously thickened, deep plum-red in/

in colour, and the mucosa may resemble the surface of a large granulating wound. A case of this in-:tensity is more likely to be met with in patients who happen to be affected with renal disease of long standing, and death takes place from poisoning before the changes later to be described have had time to occur.

If the patient survives this stage, the superficial layers of the entire mucosa undergo coagulation necrosis, become bile-stained, and are replaced by a green diphtheritic false membrane which may entirely hide the red granular layer under-:neath it. Here or there smaller or larger areas of this membrane may have sloughed, disclosing buds of granulation tissue of greater or lesser extent. Coincidently, small but numerous intramucosal haemorrhagic extravasations take place.

If the patient again survives this

stage/

stage small superficial ulcers, affecting particular-:ly the summits of the valvulae conniventes and mucosal folds, are formed. The minutely eroded, pinkish-grey "coraline" appearance of these ulcers, which run transversely across the bowel along the ridges of the mucosal folds of the colon, and are often present in large numbers, confers a character-:istic appearance. The sloughed mucosa is gradually replaced by well-formed and vascularised granulation tissue, and later by a fibrous hyperplasia; if the patient recovers entirely the large intestine becomes shrunken into a thick-walled firm tube, with narrowing of its lumen and permanent damage to its functions.

Again, the ulcerative process may continue and spread, with the result that the gut wall may be honeycombed with myriads of small ulcers and so finally resemble a piece of red porous rubber./

rubber.

These ulcers, in spite of the fact that they may give rise to the so-called "worm-eaten condition", can easily be distinguished from the amoebic ulcers by the fact that they are more super-:ficial, more regularly circular or oval, and have cleaner cut edges than the ragged undermined amoebic ulcers. Their depth will vary with the amount of fibrous change that has taken place in the bowel wall around them, which may be so great as to make them appear deeper than they really are. Whereas the thickened peritoneum may often constitute the base of of a healed amoebic ulcer, it is extremely rare to find a bacillary ulcer penetrating deeper than the muscularis mucosae. Perforation or peritonitis is practically unknown in pure bacillary dysentery.

Varying with the intensity of the infection, certain areas of the intestine are affect-:ed/

affected more than others.

Rogers states that the descending colon and rectum are more frequently involved than any other part. In our experience, on the other hand, the caecum, first part of the ascending colon, and the flexures have been more profoundly affected than the other parts of the large gut.

When complete recovery has occurred after a less extensive infection than that first described the mucosa is often seen to be atrophied and stained a dirty slate-grey colour as a result of the diffuse intramucosal haemorrhagic extravasation. In healed amoebic ulcers, on the other hand, this staining is seen only in and immediately around the cicatrices of the ulcers.

Concurrently with the local changes are/

are those produced in heart, liver, kidneys, adrenals, and probably the nervous system, due to the absorption of the toxins formed in the gut by the dysentery bacilli.

In bacillary dysentery, as in diphtheria, the patient dies of intoxication. It may be definitely stated that as a general rule dysentery bacilli do not invade the blood stream, except during the stage of premonitory fever before diarrhoea begins. On one or two occasions certain observers have claimed to have isolated them by blood culture; but this invasion of the blood must be looked on as an accident in the course of the disease, and the occasions on which a general dysenteric bacillaemia takes place are so rare that the probability of its occurrence is negligible.

Just as the amoeba is responsible for the ulcers of amoebic dysentery, so is the dysentery bacillus/

bacillus accountable for the damage to gut and general system. We do not deny that secondary infection of the blood stream may and does take place occasionally in the course of both diseases, but we controvert the idea that any such infection is to be regarded as a regular concomitant.

It has been known for many years - in fact, since Todd first worked out the poison of Shiga's bacillus - that the "non-manite-ferment-:ing" type of dysentery bacillus produces a soluble toxin. Whether this toxin is really extracellular and diffusible from the first or whether it becomes so owing to a rapid breaking down of the bacillary bodies, and consequent liberation of endotoxin is a matter of no moment. The fact remains that a filtrable toxin is easily demonstrated in cultures of this bacillus.

Everyone who has had experience of dysentery/

dysentery due to the mannite-fermenting type of bacilli must have been forced to the conclusion that a similar and equally virulent toxin was at work in these cases, as in those in which Shiga's bacillus was the morbific agent. The clinical symptoms are the same, and the degenerative changes in the organs seen at autopsy are indistinguishable from those seen in cases of death from Shiga's bacillus.

DIAGNOSIS BASED ON THE CYTOLOGICAL PICTURE OF THE STOOLS.

It will thus be seen that it is easy to diagnose between the two great types of dysentery when/

when they reach the post-mortem table, so definite and peculiar to each is its morbid anatomy. It was hoped, therefore, that on a priori grounds the differences in the nature exudate and desquamated cells found in the stools of each variety would be sufficiently constant and characteristic to constitute a basis for a more rapid differential diagnosis, and investigations both in our laboratory and in others throughout Egypt have led us to the conclusion that it is possible to arrive at such a diagnosis, based on the cytological picture peculiar to each type of the disease.

The character of any given stool will depend upon the particular stage of the disease at which that stool has been evacuated. It will depend to a much less extent upon the nature of the food which the patient has taken, in that in the vast majority of cases both types of patients - amoebic and bacillary - will have been treated on similar dietetic/

dietetic lines; further, our examination is re-
:stricted to any sloughs, bloody mucus, etc., which may be mixed with the faecal matter.

A. CYTO-DIAGNOSIS IN AMOEBIC DYSENTERY.

If some of the blood-stained mucus from a case of "simple" amoebic dysentery be examined microscopically the first thing noticable will be the scantiness of the cellular exudate, although red blood corpuscles may be very abundant. This point has particularly impressed us when examining material taken/

taken from the surface of an ulcer at autopsy.

Another salient characteristic is the preponderance of mononuclear cells over polymorpho-nuclears, a feature which contrasts sharply with the picture seen in bacillary dysentery.

Next to the scantiness of the cellular exudate the most noteworthy element in the film is the picture presented by the actual cell components - most of them look half-digested, and have a "mouse-eaten" appearance. The process evidently begins at the periphery, and the nucleus is the last to be involved. In many cases groups of nuclei appear to become agglutinated by means of their semi-digested surrounding cytoplasm. The process is apparent-:ly a slow one, the cytoplasm being gradually digested and dissolved and the unchanged nucleus being left bare or clothed merely in a narrow strip of ragged cytoplasm.

As/

As will be shown later, the cells in a bacillary stool give evidence of being poisoned *en bloc* and undergo rapidly the changes characteristic of toxic necrosis and autolysis, the nucleus being among the first parts of the cell to be affected.

Further, the fatty changes to be described as occurring invariably in cells composing the bacillary exudate are never seen to anything like the same extent in amoebic stools, when examined fresh in Sudan III. solution; all that one sees are isolated "lumps" or dots of fat, suggesting that the cells had previously ingested some fatty material rather than that it was of intrinsic cytoplastic origin.

Consequently, all the evidence goes to prove that every morbid change seen in amoebic dysentery can be explained on the hypothesis of proteolytic digestion of living tissue by the amoebae./

amoebae.

The characteristics of a "simple" amoebic stool may be summarised as follows.

1. Scantiness of cellular exudate, especially the polymorphonuclear element.

2. Preponderance of mononuclears over polymorphonuclears.

3. Evidence of proteolytic digestion of the cells, beginning at the periphery and affecting the nucleus last.

4. Absence of all phenomena characteristic of inflammatory reaction, toxic necrosis and consequent autolysis.

B./

B. CYTO-DIAGNOSIS IN BACILLARY DYSENTERY.

A very different picture is seen on examining microscopically the blood-stained muco-pus from a case of bacillary dysentery, whether in the acute, subacute, or chronic stage.

Here the first feature to strike the observer is the abundance of cellular exudate, composed largely of polymorphonuclear leucocytes. Desquamated and degenerated mucosal cells are also common, but the polymorphonuclear neutrophiles predominate. Blood may or may not be present according to the stage of the disease, whereas it is hardly ever absent from the stools of amoebic dysentery./

dysentery.

The polymorphonuclear neutrophiles show the characteristic changes consequent on toxic necrosis more than any other leucocytes, the eosinophiles less and the lymphocytes least of all. The cell is evidently poisoned *en masse*, and whether the further changes about to be described are directly caused by the toxin or are merely autolytic phenomena resulting from the death of the cell is of no moment. The The important point for our purpose is that the pro-:cess is obviously different from that which takes place in the cells of an amoebic exudate.

The nuclei seem to be specially sensitive to the action of this toxin, as the changes described, particularly fragmentation and agglutina-:tion of small chromatin masses round the nuclear membrane, and "ringing" of the nuclei, have not been observed in similarly prepared films of pus obtained from/

from other sources - e.g., boils - or at least to nothing like the same degree.

A type of cell which is always present, often in large numbers, and which seems to be associated more frequently with bacillary than with amoebic dysentery somewhat resembles a free resting amoeba, and it is important not to mistake it for one.

There is, in our opinion, little doubt that this mistake has been often made, and that as a result the numbers returned as amoebic dysentery in 1915 were unwarrantably high. Particularly do we consider these remarks applicable to cases of so-called "running amoebic dysentery", often accompanied by a rise of temperature, which responded beneficially to serum treatment. These cells may have also given rise to the diagnosis "post-amoebic colitis".

As these cells are larger than a polymorphonuclear leucocyte, and as the changes in them are much the same as those occurring in other leucocytes, it will be convenient to describe them as a type.

They are very large mononuclears, 15 to 30 in diameter, with basophilic non-granular cytoplasm, and are probably macrophages of endothelial origin.

The cytoplasm, if not too degenerated, contains numerous vacuoles in which there may be entire polymorphonuclear leucocytes, lymphocytes, red cells, or their fragments, and bacteria, more or less digested. Curiously enough, the ingested leucocytes are much less degenerated than their hosts. By Achard's method of eosin staining these large cells can usually be demonstrated to be dead. In others, as a result presumably of more advanced de-

:generation/

degeneration, the cytoplasm, instead of being granular and vacuolated, is swollen, hyaline, transparent, and apparently in a state of hydropic degeneration; it stains very poorly or not at all with iron-alum-haematoxylin.

The degenerative process begins early in the cytoplasm, which first becomes granular, then vacuolated, and finally hyaline. It may show dots of granules, which are greenish when examined in the fresh state and intensely black when stained with iron haematoxylin.

These are probably fragments extruded from the pyknotic nuclei. In fresh specimens these dots are frequently seen to exhibit distinct Brownian movements and are apt to be mistaken for bacteria or other debris which the cell has ingested. Coincidently with the cytoplasm the nuclei begin to show/

show very definite karyorrhectic and karyolytic changes, both in fresh and stained specimens.

The nucleus first becomes swollen, then granular; finally its centre becomes trans-:parent and the chromatin is aggregated under the nuclear membrane, leaving the centre bare. The nucleus then bursts, and the granules resulting from this action appear as a scattered line of irregular dots - green in the fresh and black in the stained specimens - which are apparently caught in the periplast, all that remains of the cytoplasm.

Later these dots disappear, and the cell is represented only by the periplast, which is usually regularly circular. These cells, as their suggested origin implies, possess certain feeble powers of amoeboid movement, a characteristic which is liable to entrap the unwary.

These endothelial macrophages resemble resting/

resting or dead amoebae most closely when they show the aggregation of chromatin granules scattered around the periphery of the nucleus; then one sees a large round cell, vacuolated and containing ingested cells, with a nucleus which exhibits the "ringing" so characteristic of the nucleus of amoebae. The fact, however, that the nucleus in a degenerating amoeba is the last structure to disappear may be of some guidance in differentiation.

Similar degenerative changes to those mentioned occur in the leucocytes and also to a less extent in the mucosal cells.

Fatty degeneration is always present in the cells composing the exudate in bacillary dysentery. In the very early stages this change is very slight and the intracorpuscular globules present in leucocytes and desquamated mucosal cells appear/

appear as dust-like red granules when examined under the immersion lens, ~~and a~~ when Sudan III. in alcoholic solution is run in under the coverslip.

Later these globules become larger and larger, until in the advanced cases the greater part of the cell body is filled with them. In some we have even seen the leucocytes distended with large red globules crammed together inside the periplast like potatoes in a sack. This condition is best seen in the liquid green pea soup type of stool, without obvious muco-pus or blood, characteristic of advanced subacute or chronic bacillary dysentery.

Another point to strike the observer in cases of acute bacillary dysentery is the extra-:ordinarily small number of bacteria present; the film, in fact, looks more as though it had been taken from the pus of a streptococcal abscess than from a stool.

The/

The characteristics of a bacillary stool may be summarised as follows.

1. Abundance of cellular exudate, mostly polymorphonuclear.

2. Preponderance of polymorphonuclears over mononuclears.

3. Evidence of toxic necrosis of cells, the degenerative changes occurring early in all parts of the cell, including the nucleus. This degener-:ation may go so far as to leave only the circular periplast of the cytoplasm, thus constituting the "ghost-cell", the presence of which in any quantity is very typical of bacillary infection.

4. Evidence of phenomena characteristic of intense inflammatory reaction to microbic infection.

SUMMARY AND CONCLUSIONS.

1. Pure amoebic dysentery - i.e. uncomplicated by coexistent bacillary infection - gives rise to a characteristic exudate in the stools.

2. Bacillary dysentery, whether complicated by amoebic infection or not, also gives rise to a cellular exudate of specific character in the stools.

3. The finding of Entamoeba histolytica in the midst of a "bacillary" exudate indicates, not that the case is one of simple amoebic dysentery, but that a double infection is present,/

present, although all attempts to isolate dysentery bacilli may fail - as they do in many instances even when the case is one of "simple" bacillary dysentery.

4. In such cases of double infection, which are of more frequent occurrence than has been supposed, the diagnosis can be made only by finding E. histolytica, for the reason that the exudate associated with the latter is comparatively so scanty and insignificant that it is masked by the overwhelming bacillary exudate. While the consequences of neglected amoebic infection may be disastrous, it is of still more immediate importance that bacillary dysentery should receive prompt recognition and appropriate serum treatment.

III. STUDIES ON TREATMENT.

1. TREATMENT OF AMOEBIC DYSENTERY BY EMETINE BISMUTH IODIDE.

The treatment of carriers of Entamoeba histolytica with the double iodide of emetine and bismuth was originally tried at certain convalescent hospitals in England early in 1918, and at the time when this work was carried out was more or less on its trial. It was of interest, therefore, to try to estimate its value as a curative agent in some of our cases in Egypt.

It/

It has been shown that emetine hydro-:chloride, administered hypodermically even in full doses was successful in about one-third only of the cases treated, and the following investigations, while of little more than historical interest now, at that time were of considerable significance.

To appreciate the results of treatment with this drug it must be noted that this series of cases was given emetine bismuth iodide because they had proved refractory to ordinary emetine injections, being still infected after receiving large doses of emetine hydrochloride hypodermically.

It was not understood at that time that the drug should be used in large quantities if the treatment was to be in any measure successful. One grain of emetine hydrochloride is approximately equal/

equal, in emetine content, to three grains of the double iodide, and consequently, to obtain the equivalent of a full course of twelve grains of emetine injection, some thirty-six grains of the double iodide had to be administered to the patient.

Table I shows a series of ten cases which received twenty-four grains or less of the double iodide - the equivalent of approximately eight and three-quarter grains or less of the hydrochloride.

Table I.

Case No.	Total amount of drug given in grains	Result
1	14	No effect.
2	14	Relapse after 4th day.
3/		

Table I. (contd).

Case No.	Total amount of drug given in grains.	Result
3	8	No effect.
4	14	No effect.
5	12	Relapse after 5th day.
6	8	No effect.
7	24	Relapse after 8th day.
8	6	No effect.
9	16	Relapse.
10	12	Relapse.

It will be seen that there is not a single successful treatment in the whole series, either there was no effect, or a relapse occurred in every case, and the conclusion to be drawn from these facts is obvious.

Table/

Table II shows the results of treatment carried out on twenty-four cases. Each case received altogether a minimum amount of thirty-two grains.

Table II.

Results of Treatment with Emetine Bismuth Iodide in Larger Quantities.

Case No.	Total amount of drug given in grains.	Result
1	36	Cured
2	36	Cured
3	32	Cured
4	42	? Cured
5	40	Not cured.
6	32	? Cured
7	40	Cured
8	40	Cured
9	68	Cured
10	36	Cured
11	42	Cured
12/		

Table II. (Contd).

Case No.	Total amount of drug given in grains.	Result
12	36	Cured
13	36	? Cured
14	40	Cured
15	32	? Cured
16	32	? Cured
17	36	Cured
18	36	Cured
19	36	? Not cured
20	40	? Cured
21	32	? Cured
22	36	Cured
23	36	Cured
24	32	Cured

From this Table it may easily be seen that the results were much more satisfactory than in the previous/

previous series of cases. It seemed reasonable to conclude therefore, that treatment with emetine bismuth iodide, given in sufficient quantities, was considerably successful in the treatment of carriers of amoebic dysentery.

From the small number of cases and from the varied results of other observers it was too soon to claim that this drug was a certain means of cure. But there is no doubt that it was much superior to the older treatment with emetine injections, as most of these cases had previously been given the hydrochlor-:ide, often for a prolonged period, without success.

Only two cases of acute amoebic dysentery were treated with the double iodide. One of these had previously a full course of emetine in-:jections, (one grain a day for twelve days), Both patients were rapidly and satisfactorily cured. The results/

results were promising, but unfortunately no further cases were available for trial during my time in Tel el Tebir.

CONCLUSIONS.

(1) Emetine bismuth iodide, properly administered has successfully cured the majority of carriers of E. histolytica which have hitherto been treated.

(2) For treatment to be successful, the drug must be given in large quantities - not less than 36-40 grains, in daily doses of three or four grains. Less than this amount is seldom efficacious, and more may be necessary in individual cases.

2. TREATMENT OF OTHER PROTOZOAL INFECTIONS BY EMETINE BISMUTH IODIDE.

There is little to say concerning the treatment of any protozoal infection other than that with E. histolytica. It seems highly probable that no method has yet been devised for removing any other protozoal inhabitant of the human bowel. There are, however, certain treatments which have been tried and found wanting, and which are therefore worthy of mention as they can now be definitely discarded as useless. Some of these may be briefly noted.

Entamoeba coli /

Entamoeba coli. - It is clear from all the records in which cases have been followed for any length of time that treatment with emetine hydro-:chloride hypodermically will not remove E. coli. When due regard is paid to the frequency with which negative examinations may be made on untreated persons infected with this organism it appears highly probable that hypodermic injections of emetine have no effect whatever uponit. Cases may, indeed, become negative during and after treatment; but sooner or later they become positive again: and if daily exam-:inations have been made during both treated and un-:treated periods there appears to be no significant correlation between negative examinations and treated periods.

There is, however, some evidence that emetine bismuth iodide occasionally removes E. coli. Several cases have been recorded in which treatment with/

with this drug has been followed by the complete dis-:appearance of both E. histolytica and E. coli from the stools.

Of the cases which were treated with emetine bismuth iodide for E. histolytica infection, thirteen were infected with E. coli. Of these, three were not followed for a sufficient time to determine the possible effects of the drug upon their E. coli infections. The other ten cases behaved as follows: one was positive every time he was examined both during and after treatment; eight be-:came negative during treatment and for a variable period afterwards, and then became positive again; one became negative during treatment and has remained negative ever since.

The remarkable coincidence between the negative/

negative period and the time of treatment in eight of the cases, and the apparently complete disappear-:ance after treatment in one case, lead one to sup-:pose that emetine bismuth iodide is not without effect upon E. coli. The fact that nine cases out of ten treated were however, certainly not freed from infection, leads one also to suppose that the effect is rarely permanent.

I find no evidence, based on adequate numbers of negative examinations, that any other method of treatment has any effect upon E. coli.

Lamblia. - There is a considerable amount of evidence to show that no method of treat-:ment has yet been found that will remove a Lamblia infection. It is well known, when dealing with negative/

negative examinations, that a very large number of examinations must be made before it can be said with certainty that anybody is not infected with this organism. Negative examinations are so frequent-:ly made on untreated cases that it is necessary to examine an infected person for a very long time to demonstrate that any treatment which he may undergo has any effect whatsoever upon his infection. A few negative examinations are without significance.

Many of the infected men under my observation have been treated with emetine hypodermic-:ally for E. histolytica infection. There is abundant evidence to show that this treatment is without effect on Lamblia. As there is not even one possible "cure" in any cases where numerous examinations have been made, it will be unnecessary to enter into details. Emetine bismuth iodide seems likewise without effect upon Lamblia. It thus/

thus seems clear that emetine compounds have no effect whatever upon Lamblia.

Trichomonas and Chilomastix. - There is no infection with either of these organisms, in any series, which appears to have been cured by any treatment employed. Emetine, either hypodermic-:ally or as the double iodide, has no apparent effect upon either. This seems worthy of note. But as no claims to success have been made for any treat-:ment, and as no reports deal specifically with the treatment of these infections, it will not be necessary to consider this subject here in greater detail.

CONCLUSIONS./

CONCLUSIONS.

(1) Emetine bismuth iodide may exceptionally prove successful in removing an infection with Entamoeba coli. As a rule it is not efficacious. No other drug has yet been shown to have any action upon this organism.

(2) There is no evidence, from the cases in the present series, to show that a successful method of removing flagellate infections (Lamblia, Trichomonas, Chilomastix), has yet been found.

3. THE SERUM TREATMENT OF BACILLARY DYSENTERY.

It is not necessary in this Thesis to discuss in detail the general treatment of acute bacillary dysentery, but the author's observations on the results of serum therapy may be of interest.

It was my custom to give heroic doses of serum to all new patients admitted to my wards with acute diarrhoea and blood and mucus in the stools. I did not wait for detailed pathologic-:al reports apart from an immediate stool examination, which as has been already seen, was of the greatest value/

value in clinching the diagnosis.

Sixty to eighty cubic centimetres of anti-dysenteric serum, supplied to us from the Lister Institute and prepared from the Shiga bacillus and one or more strains of Flexner bacillus, followed by one hundred and fifty to three hundred cubic centi-:metres of normal saline, was given twice daily for the first two days and once daily for the two follow-:ing days.

There is no doubt, (this was written some ten years ago), that this giving of serum was in some cases merely a ritual, as we were in the dark about the exact typing of the different strains of organism which were causal in our part of the country. But I do maintain that the life of a patient suffer-:ing from acute dysentery depended very often on the giving of serum at the earliest possible moment.

It is a practical impossibility to examine bacteriologically all cases of dysentery occurring among troops on active service, especially when they are scattered over wide tracts of country, and, further, a negative result will most probably ensue if the proper precautions are not adopted for a correct examination of the specimen.

Under the most favourable conditions a competent bacteriologist can determine whether an infection is due to the Shiga or Flexner bacillus in twelve to sixteen hours. This fact, however, is of little value under the conditions which exist with an army in the field.

If, however, every case of acute dysentery which required serum received a mixed anti-serum to begin with, and all proved Shiga cases had received anti-Shiga serum, better results might have been obtained.

During/

During the Gallipoli campaign, and in Macedonia during 1916, 1917, and 1918, the vast majority of cases of bacillary dysentery were caused by the Shiga bacillus. In our Hospital this was also our experience.

In a series of approximately two hundred consecutive cases of bacillary dysentery who received serum our death rate was 1.5 per cent.

In the vast majority of instances the cases did not arrive at the Hospital until after the third day of the disease, some patients often having twenty or more blood and mucus stools per day. These figures, therefore, are of real significance.

In the cases that responded best, the temperature fell within twelve hours of the initial dose. The toxaemic symptoms, such as collapse and prostration were satisfactorily alleviated. There is no doubt that the giving of intravenous saline/

saline was a factor of great significance in the improvement which was noticed. The distressing abdominal symptoms such as pain, tenesmus and urgent call to stool, all appeared to be more rapidly re-:moved than in our previous experience, when we had no serum available.

There is no doubt, therefore, that the early neutralisation of toxins in patients suffering from severe bacillary dysentery by the giving of a potent anti-dysenteric serum is an essential factor in the treatment of the disease.

4. CHLORINE WATER AS AN ADJUNCT IN THE TREATMENT OF DYSENTERY.

The use of chlorine water lavage was advocated many years ago by Burney Yeo in the treat-:ment of typhoid fever with satisfactory results. This idea was applied in the treatment of infantile diarrhoea, and in a paper on the subject which I published in the British Medical Journal, (15th December, 1917), it was pointed out that a large percentage of cases responded well to chlorine lavage, and it occurred to me therefore that a similar treatment, used of course in combination with emetine and serum, might be of value in cases of dysentery./

dysentery. The following results, which were embodied in a Report to the Senior Medical Officer in the Division, may therefore be of interest.

In this part of my investigation notes were made on five-hundred and two cases of dysentery, of varying severity, which received this form of treatment, in addition to the routine measures.

Of these four-hundred and four were bacillary, eighty-eight amoebic, and ten with both bacillary and amoebic.

Seventeen cases died, thirteen bacillary and four amoebic and two with both bacillary and amoebic.

Of the thirteen bacillary cases that died three were complicated with pneumonia, two with tuberculosis, and three with pellagra. Of the four cases that died from amoebic dysentery, three were/

were complicated with pulmonary disease, and one with malaria.

At the time of this special investiga-:tion there was an abnormal number of daily admissions, and most of the patients being admitted in a very severe and advanced stage the death rate was accord-:ingly higher than would have been the case if the patients had been treated at an earlier stage of the disease.

In addition to the dysentery cases one-hundred and six cases of acute diarrhoea came under observation; in fourteen of these Lamblia cysts were reported. These cases also responded to chlorine water lavage in a highly satisfactory way.

Method of Preparation of Chlorine Water for Clinical Use. Into a glass stoppered bottle put 200 grains of potassium chlorate./

chlorate. Then introduce 300 minims of hydro-:chloric acid (fort.) and allow to stand for three minutes until air is replaced by chlorine gas. Gradually add distilled water, shaking all the time, until the bulk is 1 pint. This gives approximate-:ly a 2½ per cent solution of chlorine. For clinical use the strength was then diluted to 1 per cent, and prepared fresh every day.

For the actual lavage put half a pint of 1 per cent solution in container, add an equal amount of warm water and use two to four times daily, (i.e. a 0.5 per cent solution).

Method of Administration. Take a 100 cubic centimetre glass syrine, remove piston and fit to nozzle two feet of narrow rubber tubing. Into the free end of the rubber tubing fit a No.12 or No.14 red rubber catheter, (or stomach tube connected to rubber tubing by glass nozzle of enema syringe.)/

syringe).

The catheter or stomach tube, whichever is used, is inserted into the rectum to the full length, (in the case of the stomach tube to sixteen inches), with the patient in the left lateral position or the genu-pectoral position.

The funnel is then raised and the chlorine water allowed to run in slowly until about one and a half pints have been passed into the large intestine. (After withdrawal it is necessary that the patient should rest in the prone position for at least a half to one hour.)

A mixture of chlorine water was also given by the mouth three or four times daily.

Lavage was continued three to four times daily until blood and mucus ceased appearing in the stool, and thereafter twice daily for eight to ten days. An interval of two days was then allowed/

allowed to pass, and the stool sent for bacteriologic-
:al and cytological examination.

Result of Treatment. The striking thing in both types was the marked diminution in the number of stools per day, and the early cessation of blood and mucus, usually on the third or fourth day after the administration of chlorine lavage.

After lavage it was a noticable fact that the patient always felt easier, the tormina and tenesmus being greatly alleviated.

In the very acute cases where sloughs and foul-smelling faeces were passed there was a decided improvement.

In several cases the patient found it difficult to retain the chlorine water at first, but soon overcame this difficulty and after several injections/

injections was able to retain it for an hour or even longer with no discomfort.

Of the four-hundred and four cases of BACILLARY DYSENTERY in this series, two-hundred and eighty-six were treated with both chlorine water lavage and anti-serum; fifty with chlorine water lavage only; and sixty-eight with anti-serum only. The cases were of more or less equal severity. There is absolutely no doubt that the cases who received both serum and chlorine water lavage were benefited to a much greater degree than either those who received serum, or lavage only. (See Charts in Appendix which are representative of all this series of cases).

In a series of eighty-eight cases of AMOEBIC DYSENTERY of approximately equal severity, forty patients received chlorine lavage in addition to emetine or emetine-bismuth-iodide, and the remainder received only emetine. As in the previous group of cases, the results were highly satisfactory/

satisfactory. Those who received chlorine water lavage were certainly more rapidly improved with earlier alleviation of symptoms. (See Appendix).

Post Mortem Evidence. Autopsies were performed in a number of cases. The results here were most striking. In the cases that had had chlorine water lavage the rectal and intestinal walls showed ulcerated areas which were definitely in the process of healing - evidenced by the presence of well-formed vascularised granulation tissue, and scars of recently-healed ulcers.

In those cases who received no rectal chlorine water the ulcers presented by no means such a healthy picture. The ulcerated areas were well defined, superficial, with basal patches of necrosis. The surrounding tissues showed sloughing, along with marked hyperaemia and intense intra-mucosal haemorrhagic extravasations.

There is no doubt that, even in fatal cases, the local condition in the intestinal wall is very much improved by the use of chlorine water lavage in this condition.

Lavage with other Intestinal Anti-:septics. Lavage with normal saline and potassium permanganate (1:1000, 1:2000, 1:3000 solutions) was also tried in several patients, but with no success. The clinical condition was unimproved.

SUMMARY AND CONCLUSIONS.

SUMMARY AND CONCLUSIONS.

1. A brief account is given of the historical aspect of dysentery, with regard to the clinical picture, diagnosis and treatment of the disease.

2. The comparative pathology of amoebic and bacillary dysentery is based on the author's personal examination of over twenty cadavera, and several hundreds of stools.

The value of cytology in diagnosis is discuss-:ed, and the conclusion is arrived at that differentiation between the two great types of dysentery is possible by simple stool examination. It is not claimed that this is a final means of proof, but that, during the stress of war conditions it should be employed as an adjunct in/

SERUM

A

CLINICAL CHART.

(To be attached to Case Sheet.)

Army Form B. 181.

Corps _____ Military Hospital No _____

No. 7104 Rank and Name PTE. K. E. Age 20 Service 2 YEARS.

Disease BACILLARY ... DYSENTERY Date of admission 29.12.18 Date of discharge _____ Result CURED.

Dates of Observation	29/12	30	31	1/1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28
Days of Disease	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	
Temperature Fahrenheit	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time	Time
	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.	A.M. P.M.
Pulse per Minute	103	98	112	100	108	80	110	80	92	82	86		80		86		91	108		92		80		76		74		76		72		76		78		98		70	
Respirations per Minute																															
Motions per 24 hours	+ 15	+ 12	+ 10	+ 7	+ 9	+ 10	+ 8	+ 6	M 2	M 4	M 3	M 3	+ 4	M 3	M 2	3	5	3	4	2	2	1	2	2	3	2	1	1	0	1	1

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

POSITIVE BACTERIOLOGICAL

100 cc SERUM

40 cc SERUM

40 cc SERUM

40 cc SERUM

MUCUS PRESENT

20 cc SERUM

MUCUS PRESENT

MUCUS ABSENT

NEGATIVE BACTERIOLOGICAL

NEGATIVE BACTERIOLOGICAL

(6378) Wt. W4612/P566 2,000,000 2/18 McA & W Ltd A.F.B. 181/5- (E. 2565)

Signature _____ In charge of case.

CHLORINE

CLINICAL CHART.

(To be attached to Case Sheet.)

Army Form B. 181.

Corps _____ Military Hospital No

No. 7032. Rank and Name PTE. Z. C. Age 22 Service 3 YEARS.

Disease BACILLARY ... DYSENTERY Date of admission 10. 12. 18 Date of discharge _____ Result CURED.

Dates of Observation	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1/1	2	3	4	5	6	7	8
Days of Disease	4	5	6	7	8	9	10	11	12	13	14	15	16	17																
Temperature Fahrenheit	Time A.M.P.M.																													
107°																														
106°																														
105°																														
104°																														
103°																														
102°																														
101°																														
100°																														
99°																														
98°																														
97°																														

POSITIVE BACTERIOLOGICAL

CHLORINE LAVAGE 4 TIMES DAILY. "

BLOOD CEASED "

CHLORINE LAVAGE TWICE DAILY ... MUCUS PRESENT "

CHLORINE LAVAGE TWICE DAILY ... MUCUS CEASED "

CHLORINE LAVAGE ONCE DAILY ... "

NEGATIVE BACTERIOLOGICAL

NEGATIVE BACTERIOLOGICAL

Pulse per Minute	107	92	100	70	92	80	82	68	70	78	68	72	84	76	72	76														
Respirations per Minute																														
Motions per 24 hours	+ 34	+ 20	+ 10	3 12	3 4	3 4	3 2	3 3	2	1	2	1	1	1	0	2	1	2	1	1	0	1	1	1	1	0	2	1	1

(6378) Wt. W4612/P566 2,000,000 2/18 McA & W Ltd A.F.B. 181/5 (E. 2565)

Signature _____ In charge of case.

SERUM & CHLORINE B

Army Form B 181.

CLINICAL CHART.

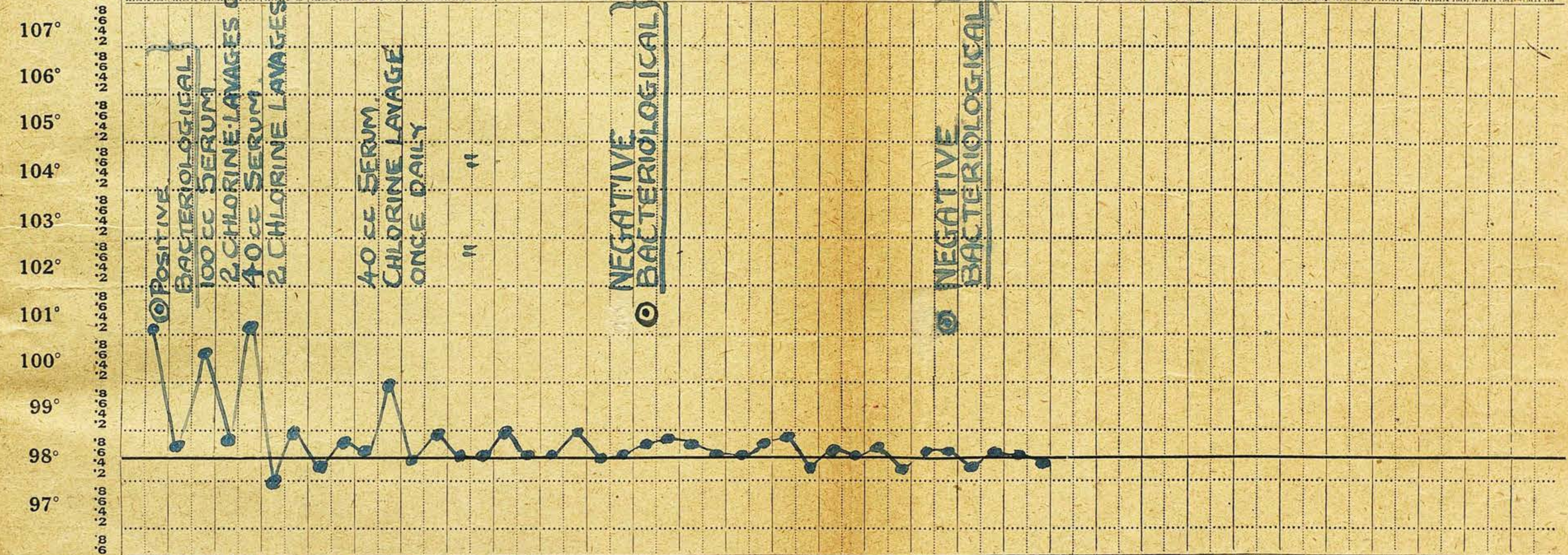
(To be attached to Case Sheet.)

Corps ______ Military Hospital No ______

No. 351 Rank and Name Pte. T. M Age 24 Service 4

Disease Bacillary Dysentery Date of admission 5·1·19 Date of discharge ______ Result Cured.

Dates of Observation	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24											
Days of Disease	6	7	8	9	10	11	12	13	14	15	16																				
Temperature Fahrenheit	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.	Time A.M. P.M.



Pulse per Minute: 108, 82, 98, 84, 112, 70, 78, 72, 92, 80, 70, 72

Respirations per Minute:

Motions per 24 hours	+ 26	+ 14	+ 6	M. 4	M. 3	M. 2	2	1	1	1	1	0	2	1	1	0	2	1	1	1

(6378) Wt. W4612/P566 2,000,000 2/18 McA & W Ltd A.F.B. 181/5. (E. 2565)

Signature ______ In charge of case.

EMETINE

CLINICAL CHART.

(To be attached to Case Sheet.)

Army Form B. 181.

Corps _____ Military Hospital No

No. 332 Rank and Name CPL. M. F Age 25 Service 3½ YEARS

Disease AMOEBIC DYSENTERY Date of admission 17.12.18 Date of discharge _____ Result CURED.

Dates of Observation	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1/1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Days of Disease	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36
Temperature Fahrenheit	Time A.M. P.M.																														

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

POSITIVE AMOEBIC BACTERIOLOGICAL ...

GR 1/3 EMETINE

GR 1 EMETINE

GR 1½ EMETINE

MUCUS STILL PRESENT GR 1 EMETINE

NEGATIVE ... BACTERIOLOGICAL

Pulse per Minute	60	65	70	68	71	72	70	68	70	82	73	68	71	84	71	74	76	80	77	82	70	76									
Respirations per Minute																															
Motions per 24 hours	+ 15	+ 15	+ 14	+ 13	1 M 10	1 M 12	1 M 10	+ 6	+ 3	+ 12	1 M 5	1 M 3	1 M 4	1 M 3	1 M 2	1 M 4	2	1 M 2	1	2	1	2	0	1	2	0	2	1	1	1	1

(6378) Wt. W4612/P566 2,000,000 2/18 McA & W Ltd A.F.B. 181/5 (E. 2565)

Signature _____ In charge of case.

EMETINE & CHLORINE

CLINICAL CHART.

(To be attached to Case Sheet.)

Army Form B. 181.

Corps

No. 311 Rank and Name PTE. T. C Age 23 Military Hospital No

Service 3 3/4 YEARS

Disease AMOEBIC .. DYSENTERY Date of admission 8.12.18 Date of discharge Result CURED.

Dates of Observation	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	1/1	2	3	4	5	6	7
Days of Disease	9	10	11	12	13	14	15	16	17	18	19	20	21	22																	
Temperature Fahrenheit	Time A.M. P.M.																														
Pulse per Minute	102	90	98	71	88	68	72	66	70		72		80		72		68		76		70		78		76		69		86		72	75	78	76	72	80		70		72																
Respirations per Minute																															
Motions per 24 hours	+ 18	+ 16	+ 7	+ 5	+ 5	M 4	M 3	2	1	3	2	2	3	2	1	1	0	1	0	2	1	0	1	1	0	1	2	0	1	1	1

POSITIVE .. BACTERIOLOGICAL

CHLORINE LAVAGE TWICE DAILY

GR 1/3 EMETINE " " " "

GR 1 EMETINE & CHLORINE LAVAGE " " " "

MUCUS CEASED

NEGATIVE .. BACTERIOLOGICAL

NEGATIVE .. BACTERIOLOGICAL

(6378) Wt. W4612/P566 2,000,000 2/18 McA & W Ltd A.F.B. 181/5 (E. 2565)

Signature In charge of case.

CHLORINE

Army Form B. 181.

CLINICAL CHART.

(To be attached to Case Sheet.)

Corps

Military Hospital No 7 P. of W.

No. Rank and Name PTE. S. W. Age 21 Service 1½

Disease LAMBLIA DYSENTERY Date of admission 27.11.18 Date of discharge Result CURED

Dates of Observation: 27 28 29 30 1/12 2 3 4 5 6 7 8 9 10 11 12 13 14 15 25/1 26 27 28

Days of Disease

Temperature Fahrenheit: Time A.M. P.M. (8 6 4 2)

107° 106° 105° 104° 103° 102° 101° 100° 99° 98° 97°

POSITIVE. LAMBLIA BACTERIOLOGICAL

4 CHLORINE LAVAGES DAILY

BLOOD CEASED

" " " " " "

MUCUS PRESENT.

2 CHLORINE LAVAGES DAILY

" " " " " "

MUCUS CEASED

CHLORINE LAVAGE ONCE DAILY

NEGATIVE. BACTERIOLOGICAL

Pulse per Minute

Respirations per Minute

Motions per 24 hours

+	+	M	M	+	M	+	+	M																	
17	20	14	13	10	9	12	8	4	3	5	3	2	2	1	0	1	2	2	1	1	1	1			

(6378) Wt. W4612/P566 2,000,000 2/18 McA & W Ltd A.F.B. 181/5 (E. 2565)

Signature In charge of case.

APPENDIX II.

COPIES OF LETTERS SENT TO THE AUTHOR.

No. 6 Prince of Wales' Hospital,
Tel el Kebir.

February 3rd, 1919.

Dear Rosebery,

At your suggestion I have been employ-:ing Chlorine Lavage in the treatment of dysentery at this Hospital for the past month. The cases certainly improved by its aid and the motions became less frequent. I regard it as a very useful adjunct in addition to the usual methods in the treatment both of Bacillary and Amoebic Dysentery.

Yours sincerely,

Alexander Evans.
Capt., R.A.M.C.

Egyptian Army Transport Corps Hospital,
Tel el Kebir,
Egypt.

February 20th, 1919.

The beneficial results obtained from using Chlorine Lavage in combination with antidysenteric serum in cases of bacillary and amoebic dysentery have been so striking that I feel I cannot let this opportunity pass without recording my thanks to Capt. Rosebery, R.A.M.C. for his kindness in demonstrating to me the method he has employed. There can be no doubt of its extreme value in all types of cases of dysentery, and I was more than gratified by the results I obtained when using the lavage treatment in a series of cases which came under my care.

E. A. P. Brock,
Capt., R.A.M.C.

No.7. Prince of Wales' Hospital,
Egyptian Expeditionary Force,
Egypt.

March 10th, 1919.

Dear Capt. Rosebery,

The many medical officers attached to my hospital have tried your chlorine water treatment for amoebic and bacillary dysenteries with control cases for the past four months. They are in absolute agreement in the benefits obtained. The results also observed by myself have been striking and satisfactory. The absorption of toxins is lessened and both pulse and temperature have improved, also a diminution of the frequency of the stools.

I am notifying the D.A.D.M.S. of this treatment, with a recommendation for its adoption in all cases of dysentery.

Yours faithfully,

P. J. Burgess,

Col., R.A.M.C.